Case report

Pneumonitis following diesel fuel siphonage

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ABSTRACT

Petroleum diesel is a complex mixture of liquid hydrocarbons and mainly used as fuel in transport vehicles. The practice of manual siphoning of diesel from fuel tanks is common in developing countries but hydrocarbon pneumonitis due to diesel siphonage is rarely reported. We report pneumonitis following diesel fuel siphonage in a 30-year-old driver. Initially patient had severe nausea and vomiting followed by chest pain and breathlessness after three days. In our case, induced sputum was diagnostic as against majority of cases where bronchoscopy was used as diagnostic tool. Recovery was complete with medical treatment.

1. Introduction

Petroleum diesel is a complex mixture of saturated and aromatic hydrocarbons produced from fractional distillation of crude oil with chemical additives like detergents, smoke suppressants, flow improvers etc. Aspiration of diesel may occur either directly or through aspiration of vomitus secondary to its ingestion.1 Hydrocarbon pneumonitis is an acute intense pneumonitis and most patients recover without any significant pulmonary sequelae.2 So far, very few studies on hydrocarbon pneumonitis due to diesel fuel aspiration have been reported in literature.3,4 We report a rare case of pneumonitis following diesel fuel aspiration.

2. Case report

A 30-year-old tractor driver presented with right sided pleuritic chest pain and breathlessness of three days duration. There was no cough or wheezing. Three days before, he had aspirated diesel while siphoning it from the fuel tank and had cough lasting for less than a minute. Later developed nausea, vomiting and fever which subsided with symptomatic treatment at local place. On physical examination, patient was dyspnoeic but there was no cyanosis or peripheral oedema. His pulse rate was 116 beats/min, respiratory rate was 30 breaths/min, blood pressure was 96/60 mmHg and room air oxygen saturation was 86%. Chest examination revealed scattered inspiratory crackles over left hemithorax. Other systems were clinically normal. Patient was admitted with a provisional diagnosis of diesel induced pneumonitis. The arterial blood gas analysis at room air revealed a pH of 7.42; PaO2 of 60 mmHg; PaCO2 of 33 mmHg and HCO3 of 21.4 meq/L. Blood examination revealed haemoglobin of 13.4 g/dl, total leucocyte count of 11,400/cu mm with a differential of 64% polymorphonuclear leucocytes and 22% lymphocytes. His blood chemistry was normal. The posteroanterior chest radiograph done on the day of diesel aspiration revealed bilateral patchy opacities (Fig. 1) and repeat chest radiograph one week later in our hospital showed partial clearance of lung opacities (Fig. 2). Cardiac evaluation was normal. High resolution computed tomographic (HRCT) scan of chest showed bilateral patchy areas of consolidation (Fig. 3A and B). Patient declined to undergo flexible bronchoscopy and sputum was induced through nebulized hypertonic saline inhalation. The smears and bacterial cultures of induced sputum were negative. Cytological examination of induced sputum revealed foamy macrophages establishing the diagnosis of hydrocarbon pneumonitis (Fig. 4A and B). At admission, patient required supplemental oxygen for few hours and analgesics for one day. A five day course of amoxicillin-clavulanic acid and methyl
prednisolone was also given. Patient recovered quickly and was discharged after five days.

3. Discussion

After aspiration, hydrocarbons do not get absorbed in the airways and reach alveoli rapidly without evoking cough. In alveoli, they induce bronchial oedema, tissue damage and surfactant destruction. These pathologic changes result from inflammatory reaction due to activation of macrophages and release of inflammatory cytokines. All signs of activation of macrophages may be seen through electron microscopy. The host reaction to the inhaled lipid substances differ according to their chemical characteristics and manifest with mild to severe illness; sometimes leading to death. The symptoms of acute hydrocarbon pneumonitis are non-specific. The typical clinical manifestations of acute exogenous lipid pneumonia include breathlessness, cough and low grade fever which usually resolve with supportive treatment. In our case, chest pain, breathlessness were predominant respiratory symptoms. The activated macrophages phagocytose the emulsified lipid in the alveoli and detection of these lipid containing cells or foamy cells through appropriate staining techniques is diagnostic of lipid pneumonia. In majority cases of diesel induced pneumonitis, the diagnosis was made through bronchoscopic specimens. In hydrocarbon pneumonitis, bronchoscopy is useful for obtaining specimens from the site of disease and to view the inflammatory changes. In our case, we could not obtain consent for bronchoscopy and relied on non-invasive diagnostic technique like induced sputum. To our knowledge, induced sputum as a diagnostic tool in diesel induced hydrocarbon pneumonitis has not been reported so far in English literature. Chest CT features of hydrocarbon pneumonitis after diesel siphonage is rarely documented and most cases show bilateral necrotic air-space consolidation predominantly involving the right middle lobe. HRCT of chest is the imaging technique of choice as it may show typical appearances of exogenous lipid pneumonia like consolidation of low attenuation with 'crazy paving' pattern. In our patient, the HRCT of chest showed areas of ground glass appearance, bilateral patchy consolidation predominantly involving the lingula and right middle lobe without negative attenuation. Resolution of radiologic opacities following clinical recovery usually occurs between two weeks to 8 months. The short course of illness in our case could be due to the fact that only small volume of diesel could be aspirated during siphoning. We used antibiotic and corticosteroid drugs as recommended before even though their use in similar situation remains
controversial. In conclusion, when spontaneous sputum or flexible bronchoscopy is not possible, induced sputum may be an effective early diagnostic tool of hydrocarbon pneumonitis.

**Conflict of interest**

All the authors do not have any conflict of interest to declare with regard to contents of the manuscript.

**References**